REMARKS

This application has been carefully studied and amended in view of the Office Action dated December 13, 2007. Reconsideration of that action is requested in view of the following.

It is respectfully submitted that parent claim 7 and its dependent claims 3-4, 8 and 18 are patentable over Petzelt, et al. in view of Thomas and Zapol, et al. Parent claim 7 has now been amended to define the upper limit of the xenon administered to the patient as being no more than 60% and the upper limit of the xenon in the combined gas supplied to the patient as being 60% with a lower limit of 5%. There is ample support in the specification for this amendment. See, for example, page 3, line 3.

The position taken by the Examiner seems to be that since the methods of Petzelt et al. and the methods of Zapol et al. or Thomas et al. are "directed to the same purpose it would have been obvious to combine xenon and NO especially in view of the fact that Petzelt et al. suggests other gases and Thomas et al. and Zapol et al. teach using NO sources for treating the same conditions". This statement has obviously been made under the assumption that the general teaching of craniocerebral trauma in Petzelt et al. would encompass spasms such as cerebral vasospasms (see page 6, last paragraph of December 13, 2007 Office Action).

This, however, is not correct.

A craniocerebral trauma is a condition resulting from traumatic injuries involving the cranium and intracranial structures of the brain. Such injuries may be classified by whether or not the skull is penetrated (i.e. penetrating vs. nonpenetrating). The person skilled in the art, however, does not necessarily consider cerebral vasospasms as a condition that can be treated with xenon when being taught by Petzelt et al. that xenon can effectively be used to treat craniocerebral trauma. A patient can suffer from a craniocerebral trauma without ever developing any cerebral vasospasms. And, on the contrary, a patient can suffer from cerebral vasospasms without having any craniocerebral trauma. It should also be noted that Petzelt et al.

only teaches the person skilled in the art that neurointoxications caused by dopamine, glutamate and/or noradrenalin may be treated with xenon since xenon suppresses the release of these neurotransmitters.

However, cerebral vasospasms are not caused by an excess of release of any of these neurotransmitters.

It is therefore not at all obvious for the person skilled in the art to assume that xenon can be used to treat spasms, especially cerebral vasospasms when learning from Petzelt et al. that xenon can be used to suppress the release of dopamine, glutamate and/or noradrenalin. An increased release of dopamine, glutamate and/or noradrenalin, which is necessarily related to the conditions mentioned in Petzelt et al., has nothing to do with cerebral vasospasms.

Accordingly, Petzelt et al. on the one side and Thomas et al. or Zapol et al. on the other side do not describe methods directed to the same purpose. Therefore, it is not evident at all why the person skilled in the art should combine the teaching of these documents.

However, even if, hypothetically, the person skilled in the art would combine the teaching of Petzelt et al. with the teaching of Thomas et al. or Zapol et al., such person would not come to a method as claimed now in parent claim 7.

According to claim 7 as amended, xenon is used in concentrations of not more than 60% to treat spasms. Petzelt et al. does not teach that craniocerebral trauma, which according to the Examiner encompasses spasms, with xenon in concentrations below 60% by volume. With respect to the xenon concentrations, Petzelt et al. makes the following statement on page 8, second paragraph:

> "In the case of acute threatening states, such as a craniocerebral trauma or an ischemia, the respiration can advantageously be carried out with a xenon-oxygen mixture of 90:10% by volume, preferably 80:20% by volume, most preferably 75-70:25-30% by volume, over several hours to one day. As compared thereto, the

> intermittent respiration by a xenon-air mixture to which less xenon has been added, e.g. 5 to 30% xenon, preferably 10 to 20% xenon, can be considered in chronic progressions of a disease."

Thus, Petzelt et al. differentiates between the concentrations in which xenon has to be administered in the case of acute threatening states and in the case of "chronic progressions of a disease". However, the expression "chronic progressions of a disease" is only used in combination with chronic Parkinson's disease. Thus, concentrations of xenon below 70% by volume are only taught for conditions like Parkinson's disease, but not for conditions like craniocerebral trauma, which the Examiner links to spasms.

Thus, even if the teaching of Petzelt et al. with the teaching of Thomas et al. or Zapol et al. would have been combined, such hypothetical combination would not result in the method as now claimed in parent claim 7.

In view of the above remarks and amendments this application should be passed to issue.

Respectfully submitted,

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